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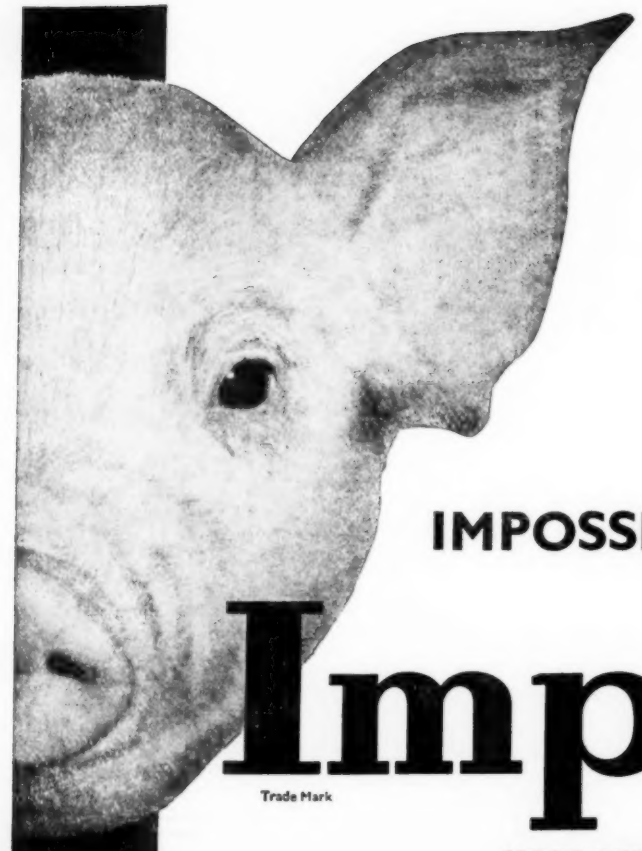

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Acute Circulatory Failure in a Herd of Pigs

BY

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SUMMARY.—A herd of pigs is described in which acute and fatal heart failure became a problem among the fattening stock. No errors of nutrition or management could be found that might have given rise to the disease.

Introduction

THE condition of acute heart failure in pigs, known as Herztod or cardiac syncope, has been recognised on the Continent for many years. More recently, Lamont, Luke & Gordon (1950) in Northern Ireland, and Ludvigsen (1953, 1954) in Denmark have described similar conditions in pigs under the titles of Mulberry Heart and Muscular Degeneration, respectively. The relationship between these diseases is not clear, mainly because the aetiology in each case is obscure, but there is sufficient similarity in their pathology to suggest that they may be either a single entity or a complex of varying aetiology that is divided unnaturally by the present pathological terms. In the herd now described, acute and fatal heart failure became a serious problem and whereas at the outset deaths occurred in the pigs approaching bacon weight, the subsequent mortality was almost confined to the young stores, as in the account of Lamont *et al.* (1950).

Clinical History

A herd of approximately 70 breeding sows was kept on one farm and from here weaner pigs were moved to a second farm where they were fattened in large straw yards. The sows were either Large Blacks, Landrace, or the first cross of these two breeds; two Landrace boars were used for service. Once the pigs were in large groups in the fattening yards they could not be individually identified and this fact made it difficult to define the conditions that predisposed to the disease. It is not clear, for example, whether certain breed crosses were more liable to be affected or whether the disease was more severe in the more rapidly growing animals.

Affected pigs commonly scoured; the diarrhoea was yellow but not profuse. There was no obvious loss of condition and no pyrexia. In general, although a number of animals might be observed to be affected in any one yard, it was not often clear which pig would die next. Thus sometimes a pig would collapse and seem about to die, but the next day it could not be singled out from the other pigs in the yard. Alternatively, a pig that appeared fairly well might suddenly collapse and die while feeding. Such sudden deaths were commonly precipitated by moving the pigs, as when weighing them, and at one time all movements had to be strictly curtailed until the worst affected groups could be sent for slaughter. The first sign of impending collapse was a slight lethargy and, as the bodily condition remained good, it was necessary to observe each group of pigs carefully for some time to see how many animals were affected. The lethargy frequently progressed to a more marked disinclination to move, when the head drooped and the ears sometimes showed cyanosis. Pigs standing in this dejected posture showed muscular shivering, particularly at the shoulders. Even so, several animals in this condition appeared to improve clinically in a day or so. More severely affected cases would not rise, but apart from cyanosis they showed little other external evidence of ill-health.

The first death that was probably attributable to this disease occurred on December 12th, 1956, when a pig weighing about 170 lb. died shortly after seeming dejected and subdued. A second pig of the same weight died on December 26th, and 4 other animals between 130 and 200 lb. at this time showed periods of lethargy alternating with improve-

ment. Three of these pigs died shortly afterwards and the practitioner who made the *post-mortem* examinations observed marked haemorrhages in the myocardium. On January 1st, 30 pigs were sent for slaughter; swine fever was suspected among them after death and the diagnosis was confirmed officially. The herd had not been vaccinated against swine fever.

As a result of the diagnosis of swine fever, 188 fat pigs were sent to slaughter and the 87 that remained were vaccinated with crystal violet vaccine on January 4th. Sudden deaths continued to occur among the bigger pigs, however, and when 8 more had died, the writer was consulted. This eighth carcase was examined on the farm on March 1st, and, as with a pig that died the following day, it showed pathological changes typical of Herztod. No changes suggestive of swine fever were present and there was no clinical evidence of swine fever among the pigs still on the farm.

As a number of other animals showed early clinical signs of circulatory embarrassment, all pigs over 110 lb. were sent for slaughter on March 5th. In all, 100 pigs were killed and their hearts were examined shortly after removal. Five hearts were very obviously affected with gross haemorrhage and these probably came from pigs weighing about 140 to 150 lb. Seven other hearts showed varying degrees of change. It is of interest that when the pigs were loaded into the transport, 4 animals were observed to be on the brink of collapse, a fact that was not known to the writer at the time. The subsequent events can be considered more conveniently with the dietary history.

Nutrition. At the time of the first deaths there were two rations, depending on the average weight of the pigs in each yard (Table I). It will be seen

TABLE I
RATIONS FED IN STRAW YARDS

Constituent	Ration until average weight 110 lb.	Ration from 110 lb. until slaughter
Barley meal	3½ cwt.	5½ cwt.
Wheat or maize	2 cwt.	2 cwt.
Middlings	2½ cwt.	2½ cwt.
Fish meal	2 stone	—
Dec. ground nut	½ cwt.	—
Dried grass	½ cwt.	—
Minerals	1½ stone	1½ stone
Vitamins (A + D_3)	+	—

that about 2.5 per cent. fish meal was fed until an average weight of 110 lb. was reached and, thereafter, the fish meal was omitted. The owner believed that a similar disease that had occurred on the farm before was related to the feeding of fish meal and, consequently, he had reduced the concentration of this constituent from 7.5 to about 2.5 per cent. 2 years previously. However, the latter concentration was now giving a very unsatisfactory rate of growth and on May 29th, 1957, the fish meal was restored to 7.5 per cent. The food conversion ratio improved from 4.0 to 3.34 but, within a few days, scouring became acute and on June 17th, two deaths occurred. Pigs continued to die until July 26th (Table II) and whereas the original deaths had been mainly among heavy pigs (115 to 170 lb.), the mortality was now largely in pigs of less than 100 lb. In an attempt to prevent further deaths, therefore, the fish meal was removed from the diet of the

TABLE II
SECOND PERIOD OF MORTALITY
The mortality rate and the weight at death during the second phase of sudden deaths.

Date	Yard	Weight at death	Remarks
		lb.	
June 17	5	74	
,, 17	5	45	
,, 24	6	90	Field *post-mortem* exam.; typical disease
,, 28	6	100	
,, 29	3	105	
,, 30	1	60	
July 11	4	90	
,, 14	4	60	
,, 17	—	—	Two pigs died; weights not recorded.
,, 21	1	100	Field *post-mortem* exam.; typical disease.
,, 21	1	120	
,, 24	1	60	*Post-mortem* exam. in laboratory; both typical disease.
,, 26	1	50	

pigs in two of the yards on July 4th. As this appeared beneficial, and as the disturbance involved in weighing other pigs on July 11th indicated that a number of animals were approaching heart failure, the feeding of fish meal was discontinued generally. Thereafter, only one more death occurred, on August 17th, until all the 115 survivors were slaughtered on September 16th. Since this date, fish meal has been fed at a level of 2.5 per cent. and the disease has not recurred.

Gross Pathology

The carcases were in good bodily condition. The skin, particularly on the abdomen, in the neck region and at the ear tips was cyanotic. The skeletal muscles were pale and very moist; on incision, the vessels exuded unclotted, venous blood.

The peritoneal cavity contained a large volume of straw-coloured fluid, in which there were strands of fibrin. Fibrinous strands were also present on the viscera and formed fragile adhesions between the organs. The livers were enlarged and had rounded edges; they were mottled, with a classical nutmeg appearance. The spleens were larger than normal, dark and turgid; on incision, they were fibrous and oozed venous blood freely. Passive venous congestion was also evident in the kidneys, in that they were dark and had a rubbery consistency. The gastric and mesenteric veins were prominent and the mesenteric lymph nodes were enlarged.

The chest cavity contained slightly blood-stained fluid, with flakes of fibrin, and the ventral parts of the lungs were collapsed. The lungs were oedematous and dark and, when cut, exuded a dark, frothy fluid. The interlobular septa were often markedly distended with a gelatinous material.

THE VETERINARY RECORD, November 1st, 1958

R. F. W. GOODWIN.—ACUTE CIRCULATORY FAILURE IN A HERD OF PIGS

FIG. 1.—Heart from pig aged 20 weeks. Note the cardiac haemorrhage, excess pericardial fluid and pulmonary oedema.

The heart and pericardium showed striking changes. The pericardium was grossly distended with a gelatinous fluid containing fibrin. The epicardium was streaked with haemorrhages, varying in size from small petechiae to large blotches a few centimetres in diameter (Fig. 1). Many of these haemorrhages were linear in distribution and followed the course of the vessels. Similar haemorrhages were present beneath the endocardium and on the interventricular septum. Large blood clots were present in all the heart chambers. Between the dark-red haemorrhages the myocardium was paler than usual. The heart valves and the great vessels were normal.

Discussion

The clinical signs and the pathology of Herztod have been well described by Pallaske (1938) and Cohrs (1940, 1942). The disease occurs in fattening pigs from about 100 lb. onwards and produces sudden death, particularly after violent exertion, such as during vaccination or transportation. At *post-mortem* examination there is general venous congestion and pulmonary oedema. Haemorrhages occur beneath the epicardium, the papillary muscles and in the interventricular septum, and these result in a mottled heart, although sometimes the heart may appear normal. Ludvigsen (1954) found similar changes in Muscular Degeneration. Mulberry Heart, described by Lamont *et al.* (1950), was also characterised by sudden death, and the *post-mortem* findings included myocardial and endocardial haemorrhages. The disease now described, therefore, has much in common with these three conditions.

There are many widely different views on the aetiology of Herztod (see Glässer, 1953) but a common theory supposes that the disease is nutritional in origin, particularly when the diet is poor Here, however, the diet was not obviously inadequate; neither was any other error of management observed. In these circumstances it is difficult to offer preventive advice and in Denmark, where the corresponding disease is often a problem, it is likewise not clear why certain herds become affected (Ludvigsen, 1957). In the herd investigated, several deaths appeared to be precipitated by a sudden increase in the amount of fish meal fed and, conversely, omitting the fish meal seemingly checked the mortality. It is probable, however, that this dietary increase aggravated a pre-existing condition, the cause of which is not apparent from the other information available.

Acknowledgments.—Some of the *post-mortem* examinations were made by Mr. A. R. Jennings.

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Clinical Note

A SIMPLE MOUTH GAG AND STOMACH TUBE GUIDE FOR RUMINANTS

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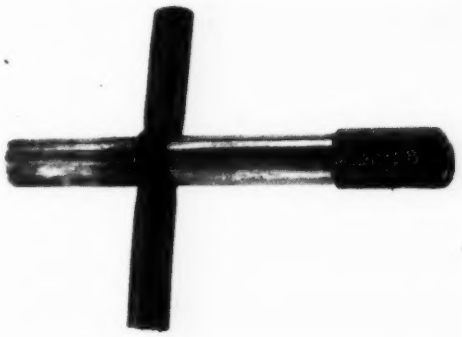
THE following is a description of a simple, cheap gag designed to facilitate the passing of a stomach tube in cattle and other ruminants. The gag (see illustration) consists of a solid rubber cross-bar, through which is inserted at right angles a hollow metal tube. One end of the metal tube is cut at an angle of 60° to the long axis and is also surrounded by a short piece of heavy rubber tubing which is cut at a similar angle and projects about $\frac{1}{8}$ inch beyond the end of the metal.



The gag is inserted into the mouth over the tongue, with the bevelled end of the tube facing upwards. The position of the rubber cross-bar should be adjusted so that it fits in the commissures of the lips whilst the bevelled tip of the metal tube approximates the entrance to the pharynx. The angled tip of the metal tube facilitates the direction of the stomach tube along the roof of the pharynx and into the oesophagus, whilst the rubber covering prevents accidental damage to the pharynx and surrounding structures. The gag is not greatly resented and the stomach tube is passed, using the minimum of force, through the metal tube into the pharynx and then on into the oesophagus when the animal swallows.

When used with cattle, an assistant holds the gag in position by grasping the protruding ends of the rubber cross-bar but with sheep the operation can be conducted by one person who holds the mouth closed over the gag with one hand and passes the stomach tube with the other.

The chief advantage of the gag, apart from its simplicity and cheapness, is the elimination of troublesome interference by the dorsum of the tongue. In consequence, a relatively rigid, thick-

(*Concluded at foot of col.* 1 *overleaf*)

Refresher Article No. 11

Anaesthetic Accidents and Emergencies

BY

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SUMMARY.—This article was written at the request of the Editorial Committee of THE VETERINARY RECORD. *It takes the form of a very brief account of some of the more common accidents and emergencies associated with anaesthesia, and indicates how they may be either avoided or treated when they arise.*

DURING anaesthesia many accidents occur suddenly. Seldom is there time to assess carefully the clinical condition of the animal, and the reason for the mishap must be recognised immediately so that the appropriate remedy may be applied at once. Other accidents are less dramatic and their results may only become apparent in the post-operative period. It is important that every anaesthetist should be aware of the nature of the more common anaesthetic mishaps since, with foresight, most accidents can be prevented.

Although one kind of emergency may give rise to another, in this article they will, for convenience, be considered separately under various headings. An attempt will be made to review their causes and to indicate how they may be prevented or treated.

Failure of the Circulation Under General Anaesthesia

Failure of the circulation under general anaesthesia is usually due to the loss of blood. There may be either a sudden effusion of blood or, more often, an almost imperceptible loss which occurs during the course of a long operation. Unless blood loss is actually measured it is very difficult to estimate the amount of haemorrhage caused by operation or trauma. It would be very interesting to know how much blood is shed during veterinary operations and how often these amounts of blood are immediately replaced, even in part. In an emergency due to the loss of blood, compatible whole blood, plasma or plasma volume expander must be given rapidly and the quantity and speed of replacement are the factors which determine the fate of the patient. Dextran solutions which are often used in veterinary practice as plasma volume expanders should always be used with caution, because the infusion of large quantities of dextran solution results in a failure of the blood clotting mechanism.

Elderly animals, and animals which have lost fluid due to their pre-operative condition do not tolerate haemorrhage well. In these animals an apparently moderate blood loss may result in circulatory failure, and fluid replacement must be accurate if they are to survive even moderately severe operations.

A sudden fall in blood pressure during operation sometimes occurs without warning in an animal whose cardiovascular system is apparently healthy and where there has been but little loss of blood. The pulse becomes imperceptible, respiration ceases and the veins (noticeably in the tongue) are dilated. The pupils remain normal in size and this may be the only indication that the heart has not stopped beating. This rather alarming reaction appears to be initiated by certain surgical manipulations. For example, it may be seen during caesarean hysterotomies in cattle and sheep when traction is exerted on the broad ligament of the uterus or on the mesovarium. It may also be seen in dogs when swabs or retractors are allowed to press upon the coeliac plexus, or when the stomach and liver are manipulated. When it arises the surgeon should stop and not recommence operating until recovery has occurred. The reaction may be avoided by

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A Simple Mouth Gag and Stomach Tube Guide for Ruminants—*Concluded.*

walled stomach tube is unnecessary and a tube with a thinner wall and larger lumen may be used. This is particularly useful when obtaining rumen contents for analysis since a tube with a large diameter enables more nearly representative samples to be obtained in a shorter period of time. Using a rotary vacuum pump, supplying a vacuum of 12 to 15 inch Hg. and ¾ inch rubber hosepipe as the stomach tube, we have sampled the rumen contents of steers several hundred times without mishap.

The dimensions of the gag are not critical but must be related to the size of the animal and the stomach tube to be used. The one in use here for cattle consists of a 13-inch length of 1½-inch diameter steel conduit, on to the bevelled end of which is stretched a 2-inch length of 1-inch diameter thick rubber tubing. The cross-bar consists of a 10-inch length of very heavy walled pressure tubing having an outside diameter of about 1¼ inches. A ⅞-inch diameter hole was made through the middle of this with a cork borer and the metal tubing forced through.

A satisfactory gag for sheep has been made from an 8-inch length of ¾-inch copper conduit forced through a 5-inch cross-bar of ordinary laboratory pressure tubing.

gentle surgery, and the anaesthetist should note that gentle surgery is possible only when the patient's muscles are adequately relaxed.

Disturbances of Cardiac Rhythm

Tachycardia is usual in young animals, but in adults the pulse rate increases in shock or after the administration of such drugs as atropine. It must be emphasised, too, that in cases where relaxant drugs are being used a tachycardia may indicate insufficient depth of anaesthesia.

Cardiac arrhythmias are common in all kinds of animal under all forms of anaesthesia but they are frequently unrecognised since unless persistent gross irregularities of the peripheral pulse occur the anaesthetist is not likely to be aware of the disturbance. Electrocardiograms of animals under general anaesthesia often show arrhythmias while on clinical examination the animal appears normal.

The origin of these disturbances of cardiac rhythm is still uncertain. If the anaesthetic agent is known to depress the functional capacity of the heart muscle it is natural to assume that the direct action of the drug on the myocardium is the cause, but serious arrhythmias can be caused by the action of the autonomic nerves to the heart. The nervous system is often hyperactive immediately before operation, and stimulation of the sympathetic nerves to the heart may cause ventricular ectopic beats or even ventricular fibrillation if the heart muscle is sensitised by hydrocarbon anaesthetic agents. The combination of adrenaline with chloroform, cyclopropane, trichlorethylene or ethyl chloride is known to cause serious cardiac arrhythmias, and the amount of adrenaline which may be released into the circulation when an animal is frightened is near to that necessary to produce ventricular fibrillation in the presence of these anaesthetic agents. Carbon dioxide accumulation in the body, and mild degrees of oxygen lack may cause stimulation of the sympathetic system, so arrhythmias are common when respiration is depressed or obstructed.

Treatment of cardiac arrhythmias will depend on an adequate diagnosis and unless an electrocardiograph is available, accurate diagnosis cannot be made. However, *most cardiac irregularities disappear once adequate respiratory exchange has been established.*

An unobstructed airway must be ensured and pumonary ventilation assisted by rhythmical intermittent pressure on the chest wall or reservoir bag whenever respiratory depression occurs.

Heart Failure

Heart failure due to the action of anaesthetics has been recognised for over a century but the incidence of primary cardiac failure in veterinary anaesthetic practice is unknown.

There are two distinct types of heart failure: the first is ventricular fibrillation which is comparatively uncommon. The second and more common type is ventricular asystole, frequently termed "arrest of the heart."

It has been known since 1911 that chloroform sensitises the heart so that a mechanical, chemical or endocrine stimulus not usually sufficient to cause harm produces ventricular fibrillation, and more recently it has been shown that cyclopropane, trichlorethylene and ethyl chloride have a similar action. Fibrillation is more common under conditions of oxygen lack—*e.g.* in shocked or anaemic animals.

Cardiac arrest or asystole is the type of heart failure associated with overdosage of anaesthetic agents. Gross overdosage is probably rare, but relative overdosage is more frequent—*e.g.* the use of normal doses of intravenous barbiturates in shocked animals.

If counter measures are to be effective, diagnosis of heart failure must be rapid. Respiration does not fail as soon as the circulation ceases; it continues until the respiratory centre becomes anoxic. This must be remembered and cardiac arrest should be diagnosed, even if respiratory movements are present, when the peripheral pulse is absent, the mucous membranes an ashen colour and the pupils widely dilated. If not already in place an endotracheal tube should be introduced to ensure patency of the airway, any inhalational anaesthetics discontinued, and positive pressure respiration, preferably with pure oxygen, commenced at once. Conservative treatment is useless and effective cardiac massage must be instituted immediately.

Effective cardiac massage is only possible when the heart is actually exposed by a thoracotomy incision. No time should be lost in "scrubbing up" before making an intercostal incision, and once the heart is exposed the ventricles must be squeezed rhythmically. A steep head-down position of the animal aids filling of the heart during massage. The rate of compression of the ventricles should not be too rapid, as the heart must have time to fill with blood between compressions. In small animals the ventricles can be compressed by the grasp of one hand, but in large animals both hands must be used, and in every case, care is necessary to prevent rupture of heart by the fingertips. The best sign that cardiac massage is effectively restoring the cerebral circulation is a contraction of the widely dilated pupils.

Rhythmical compression of the ventricles is an effective treatment for both fibrillation and asystole, but the operator must be prepared to continue this manoeuvre for long periods of time. Ventricular fibrillation is apparently more refractory to treatment than is asystole. However, once the massage has caused re-oxygenation of the myocardium, a spontaneous regular cardiac rhythm should reappear. An atonic heart may sometimes be stimulated to contact by the injection of drugs. For example, 1 in 10,000 adrenaline solution may be injected into the cavity of the left auricle and the heart massaged to force the solution into the aorta and through the coronary vessels. Fibrillation may sometimes be overcome by similar intracardiac injection of (a) 1 per cent. procaine hydrochloride, or (b) 4 per cent. potassium citrate followed by 10 per cent. calcium

borogluconate. Cardiac massage must, of course, be continued between injections. If the circulation it restored, the chest should not be closed until spontaneous heart beats have been present for some minutes.

It should be obvious that cardiac failure is only likely to be treated with success in the smaller animals. Prophylaxis is therefore of major importance. A stormy induction of surgical anaesthesia should always be avoided and the one common factor in many anaesthetic fatalities—hypoxia—must never be allowed to occur.

Respiratory Acidosis

Serious consequences are seen when the minute volume of respiration is decreased since this causes a diminished excretion of carbon dioxide from the lungs and therefore results in the development of a respiratory acidosis.

Respiratory acidosis is commonly seen when the total gas flow rate in a semi-closed anaesthetic circuit is too low, or when the soda-lime in an absorber is exhausted. It also occurs when the airway is obstructed or when respiratory movements are hampered by the position of the animal's body on the bed or operating table. Usually this disturbance of the acid-base balance of the blood results in but little harm if the duration of anaesthesia is short, but it may have serious effects if the anaesthetic period is prolonged. Death occurs when the *p*H of the blood falls below about 6.7.

The signs of respiratory acidosis are not always obvious, for the animal's mucous membranes are pink and its pulse is slow and of good volume. Although in the normal animal an increase in the alveolar carbon dioxide level causes a frank increase in the tidal volume it is important to note that this may not occur in the anaesthetised animal. As the concentration of carbon dioxide in the alveolar air increases the blood pressure first rises, then returns to normal and finally falls. Circulatory failure, when it occurs, is rapid and is due to heart failure.

When a respiratory acidosis has developed, the animal may collapse after the operation has finished. This is because the tidal volume increases as the animal awakens from the anaesthetic, the excess carbon dioxide is rapidly excreted and consequently the blood pressure falls.

Hypoxia

During anaesthesia, hypoxia is most commonly encountered when the oxygen content of the inspired gases is low, when the airway is obstructed, or when the respiratory muscles fail due to the use of relaxant drugs or high spinal or epidural nerve blocks. It is also seen when portions of the lung are collapsed during intrathoracic surgery.

Mild degrees of hypoxia may be very difficult to diagnose. Cyanosis is usually regarded as the commonest indication of hypoxia, but not only is it an unreliable sign, it is not always easy to detect. It will be masked by pigmentation of the skin and mucous membranes, and may not be seen in anaemic or shocked animals.

The brain cells are easily damaged by hypoxia, and prolonged coma after anaesthesia should arouse suspicion of hypoxic brain damage. To prevent the progression of cellular damage from the early stage of oedema to an irreversible condition, treatment must commence as soon as possible after the hypoxic episode. It consists of the intravenous injection of a hypertonic (50 per cent.) solution of sucrose to promote absorption of the oedema fluid. A dog may be given up to 50 ml. per hour of the sucrose solution.

Hypoxia also has the effect of increasing the extent of liver damage caused by anaesthetic drugs, while its effects on the heart have already been discussed.

Laryngeal and Bronchial Spasm

Laryngeal spasm appears to be seen more commonly than bronchial spasm but both conditions may occur together during general anaesthesia.

Laryngeal spasm can occur in all kinds of animal but is probably most frequently encountered in cats under ether anaesthesia when attempts are made to force the animal to breathe high concentrations of ether vapour before the protective laryngeal reflexes are depressed. Another common complication of ether anaesthesia in cats is laryngeal "crowing"—the "crowing" noise being caused by a partial spasm of the vocal cords due to irritation of the larynx by a blob of mucus, saliva, blood or vomit.

When laryngeal spasm is troublesome the best treatment is either to spray the larynx with a solution of local analgesic, or to administer a relaxant drug, in order to relax the spasm, and then to intubate with an endotracheal tube. Attempts at intubation without the aid of local analgesic or relaxant drugs will usually be unsuccessful and will prolong the spasm. Forcible intubation through a closed glottis may result in oedema of the larynx and necessitate subsequent tracheotomy.

Constriction of the bronchioles or "bronchial spasm" is occasionally seen in all kinds of animal, but ruminant animals appear to be particularly liable to develop this complication of general anaesthesia. During light anaesthesia it is usually initiated reflexly by stimuli from the site of operation but there is some evidence that the passage through the brain of blood deficient in oxygen and rich in carbon dioxide causes bronchoconstriction.

The first warning sign that bronchial spasm is imminent is a bout of coughing, and if an endotracheal tube is not in place the larynx closes. Complete respiratory arrest follows. The chest is rigid and the lungs cannot be inflated by pressure on a reservoir bag, nor can they be deflated by pressure applied to the chest wall. Cyanosis sets in and is soon replaced by a grey pallor. If ill, the animal may die, but usually the severe hypoxia releases the spasm and the animal gasps. The gasp is followed by normal spontaneous respiration and the animal recovers. Unfortunately bronchial spasm may recur if the stimulus responsible for the first attack is still present.

In all cases where bronchial spasm occurs the anaesthetist must ensure that the upper airway is clear and that the first gasp of the animal will be of an oxygen enriched atmosphere whenever this is possible. The intravenous injection of pethidine hydrochloride or of aminophyllin will often relieve and prevent the recurrence of bronchial spasm.

Obstruction to Respiration

Respiratory obstruction is usually due to the tongue or the epiglottis coming into contact with the posterior wall of the pharynx. This type of obstruction may be overcome by extending the head and drawing the tongue forwards out of the mouth. It should be noted that in the pig over-extension of the head will also cause respiratory obstruction.

In brachycephalic dogs respiratory obstruction may result from contact between the ventral border of the soft palate and the base of the tongue for many of these animals are almost unable to breathe through their nostrils. This type of obstruction can only be overcome by endotracheal intubation.

Large blood clots may accumulate in the pharynx after tonsillectomy, tooth extraction or endotracheal intubation *via* the nostril. These blood clots must be found and removed at the end of the operation. Animals unconscious after mouth, nose or throat operations should be placed in a position of lateral recumbency.

The fact than an endotracheal tube is in position in the trachea does not necessarily mean that the airway is clear. Endotracheal tubes may kink, particularly if they are soft, they may become blocked with mucus and in the case of cuffed tubes a faulty cuff may actually occlude the end of the tube. Obstruction may also be caused by the animal biting on the tube.

Apnoea

Apnoea during general anaesthesia is very common, and if means of carrying on artificial respiration are available, need give rise to but little concern.

The treatment of respiratory failure is simply the performance of artificial respiration until spontaneous respiration is resumed. Drugs have little place in the treatment of respiratory failure. Analeptics for respiratory depression due to overdosage of narcotics are not very satisfactory but may be of temporary value. Nikethamide or similar drugs may be given intravenously but relapse after apparently beneficial effect is common. Respiratory depression due to an overdose of opiate drugs may be antagonised by the intravenous injection of n-allylnormorphine.

Aspiration of Material from the Oesophagus and Stomach

This accident probably occurs more frequently than is commonly realised for material from the oesophagus and stomach may reach the pharynx as a result of (i) vomiting and (ii) passive regurgitation.

(i) *Vomiting.* This is an active process and occurs in light anaesthesia. It is often preceded by swallowing or "gagging" movements.

When vomiting occurs the protective mechanisms of laryngeal closure, coughing and breath-holding are present and the accident should not have serious consequences. All that is necessary is for the anaesthetist to clear the pharynx of the vomited material by swabbing or suction and to allow the animal to cough vigorously before proceeding with the further administration of the anaesthetic.

It is obvious that if anaesthetics are not given to animals whose stomachs *might* contain food then aspiration is unlikely to occur, but this is a counsel of perfection which cannot always be realised in veterinary practice. The stomach may contain material many hours after the eating of a meal, particularly if an accident has occurred in the meanwhile or if the animal has gone into labour.

(ii) *Passive regurgitation.* This is most commonly seen in ruminant animals but also occurs in horses, pigs, dogs and cats. It usually happens when the animal is in the head-down position, or lying horizontally on its side, and relaxation is induced by deep anaesthesia or the use of relaxant drugs. Under these circumstances the protective reflexes are not active and aspiration occurs all too readily.

In cases of oesophageal dilatation or obstruction there may be an accumulation of fluid in the oesophagus, while the stomach may contain fluid material if there is an obstruction of the pylorus or small intestine. In deeply anaesthetised ruminant animals any increase in intra-abdominal pressure will force fluid ingesta up the oesophagus into the pharynx, and this regurgitation is frequently seen in adult cattle when anaesthesia is induced with a "knock down" dose of thiopentone.

Often, the first sign that aspiration has occurred is the unexpected appearance of cyanosis, dyspnoea and tachycardia. Obviously the severity of the condition depends on the quantity of fluid aspirated and the extent of the lung area involved.

Immediate treatment consists of thorough aspiration of the tracheo-bronchial tree—although this is often more easily advised than performed. Oxygen should be administered and attention should be directed towards the relief of bronchiolar spasm. If, after the operation, the animal develops bronchopneumonia the appropriate treatment must be instituted.

The most certain way of preventing the aspiration of material from the oesophagus and stomach is to perform endotracheal intubation with a cuffed tube immediately anaesthesia has been induced.

Intravenous Injections

The commonest mishap associated with the performance of an intravenous injection is the accidental injection of an irritant solution such as chloral hydrate or thiopentone sodium into the

perivascular tissues. When this occurs the injected irritant solution should be diluted by the immediate injection of a large volume of isotonic sodium chloride solution. Hyaluronidase may be dissolved in the saline and this enzyme will hasten the absorption of the irritant drug. No other treatment is required.

Venous thrombosis is common after the injection of thiopentone sodium but, as it does not appear for 5 or 10 days, it may be missed unless the anaesthetist has occasion to give another injection after that time. Whenever possible, thiopentone should be used as a 2.5 per cent. solution and care should be taken to see that the venous return is not obstructed when the injection is made. Venous obstruction, by acute flexion of the elbow or unnatural positions of the limbs, will result in thiopentone being retained in the limb veins and this may give rise to thrombosis.

Local Analgesia

Toxic reactions to local analgesic drugs arise when the drugs are absorbed into the general circulation at a rate greater than that at which they can be broken down in the body. Rapid absorption occurs from any hyperaemic or inflamed tissue and the rate of absorption is increased by the use of solutions which contain spreading agents (*e.g.* hyaluronidase). Accidental intravascular injection may occur even though no blood can be aspirated into the syringe. The rate of absorption is decreased by the addition of vasoconstrictor agents to the solution.

Local analgesic drugs both stimulate and depress the activity of the central nervous system. Often the toxic effects are manifested by stimulation of one part of the brain while another part is depressed. Obviously the effects will vary according to the area of the brain which is affected. Cortical stimulation produces generalised clonic convulsions, while stimulatory effects on the medulla cause an increase in the rate of depth of respiration, tachycardia and vomiting. Typical general anaesthesia with respiratory and vasomotor depression usually follows. It is uncertain whether death is due to cardiac or to respiratory failure, but it seems probable that intravascular injection causes sudden primary cardiac failure, while rapid absorption from the tissues results in depression of the nervous system and respiratory failure.

The minimum lethal doses of the various agents for the different kinds of animal encountered in veterinary practice are apparently unknown, and it is probable that insufficient attention is given to the quantities of local analgesics injected or applied to mucous surfaces.

In every case where collapse has occurred after the use of a local analgesic artificial respiration must be commenced at once. Analeptic drugs increase the oxygen requirement of the brain and should not be given. Convulsions may be controlled by the injection of *hypnotic* doses of short or ultra-short acting barbiturates. Any fall in blood pressure due to peripheral or central vasomotor failure should be treated by the intravenous injection of vasopressor drugs such as methylamphetamine. Primary cardiac failure must be treated by cardiac massage.

Complications Associated with Spinal and Epidural Analgesia

Drugs used to produce spinal analgesia may cause a reaction which affects the meninges and nerves. Clinical signs resulting from damage to nerves appear rather rapidly after the effects of the analgesia should have passed off and the nerves and region of the spinal cord subjected to the greatest concentration of the drug show the most marked pathological changes. When the main effect is on the meninges clinical signs appear later, and the reaction to the drug takes the form of an aseptic meningitis which may be mild or severe. These complications do not appear to be due to faults in technique.

Injection of solutions of analgesic drugs into the substance of the spinal cord produces a severe myelitis and neuritis.

In man, post-lumbar-puncture headache is a well known complication, and it has been observed that sheep which have been subjected to spinal analgesia behave in a manner which suggests that they too suffer from headache. The headache is believed to be due to a low cerebrospinal fluid pressure caused by leakage of the fluid through the needle puncture in the dura. It does not occur after epidural analgesia.

Infection of the epidural space is fortunately rare, but has been reported after caudal epidural block in cattle. The prognosis appears to be better than in those cases in which the infection is within the dura for the infection usually remains localised. Strict aseptic precautions should be employed whenever a spinal or epidural block is attempted.

The rapid injection of a large volume of fluid into the epidural space may cause arching of the back and opisthotonus. This reaction is presumably due to a rapid increase in the pressure in the epidural space, and is usually of short duration. No treatment is required.

Anaesthetic Explosions and Fires

The "Report of a Working Party on Anaesthetic Explosions including a Safety Code for Equipment and Installations" (H.M.S.O. London) emphasises that one of the chief difficulties to be overcome if explosions and fires are to be avoided is the lack of any general consciousness of danger.

Probably the main cause of explosions in operating rooms is to be found in static electricity, and it is very difficult to ensure that conditions are always such that a dangerous discharge of static electricity is absolutely impossible. However, more obvious causes, such as smoking and the use of gas and electric fires in locations where inflammable anaesthetics are used, can easily be eliminated.

The Disinfection of Milking Units in the Control of Mastitis Due to *Streptococcus agalactiae*

BY

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SUMMARY.—It has been shown that, when a cow is milked by machine, material regularly passes from one teat-cup to another and occasionally from the long milk tube into the teat-cups. It is desirable to disinfect all parts of the milking unit, from the teat-cups to the long milk tube, after each cow is milked.

The distribution of Str. agalactiae *in various parts of milking units which had been used to milk infected cows was investigated.*

Various methods which can be used to disinfect milking units after milking each cow were compared. When a milking unit was contaminated with sticky, purulent material, all methods gave irregular results and often large numbers of pathogens survived. When the cow's secretion contained clots only, rinsing with running water reduced the number of Str. agalactiae *present to a comparatively low level. Once this level had been reached, further rinsing had little effect. Greater numbers of organisms survived treatment with disinfectants. The most effective of the methods tried was the circulation of water at 70° C. through the unit but even this did not effect complete disinfection.*

THE control of *Streptococcus agalactiae* infection in dairy herds may be exercised in two complementary ways. These are eradication by treatment of infected cows and milking hygiene. By reducing the rate of spread of infection the latter can materially assist the former and, should the attempt at eradication fail, milking hygiene can help to keep the infection rate at a low level.

In a previous paper (Davidson, Slavin & Stuart, 1954), an investigation into some of the factors in milking hygiene was reported. It was shown that, if a sterile milking unit together with a sterile udder cloth was used for each cow, *Str. agalactiae* infection did not readily spread in an infected herd. Under ordinary farming conditions it is not difficult to sterilise, by boiling or steaming, a batch of udder cloths for each milking. It is not economically possible, however, to provide a milking unit for each cow. A method of disinfecting a milking unit after milking each cow is needed.

This paper reports the results of an investigation into the efficacy of various methods of disinfection and compares them with the traditional one of plunging the teat-cups into a bucket of disinfectant solution. None of the methods tried achieved complete disinfection and, indeed, it is suggested that complete disinfection is not possible in the short time available in the milking shed.

Spurgeon, Harper, and Elliker (1949), Hay (1941), and Stewart *et al.* (1956) have reported trials of various methods of disinfecting teat-cups and also found that complete disinfection was not obtained. These authors, however, did not attempt to disinfect parts of the machine other than the teat-cups. In each case, they recovered organisms by swabbing the teat-cup liners

Claydon (1953a) has described a method suitable for recovering organisms from the milk tubes and claw as well as from the teat-cups.

Hoy and Taylor (reported by Oliver, 1955) found that 47 per cent. of 32 clusters were positive for *Str. agalactiae* after dipping in 800 to 1,600 p.p.m. of available chlorine for 3 to 6 minutes with the vacuum tap closed and an air-lock present in the teat-cups. When the tap was open, only 8 per cent. of 113 clusters were positive. The method of recovery of the streptococcus was not stated.

Hughes (1953) disinfected milking units after each cow was milked by filling the unit with hypochlorite solution and leaving it for, on the average, 4 to 6 minutes.

Preliminary Investigations

For the effective control of mastitis pathogens, they must be removed from or killed in those parts of the milking unit which come into contact with the teat and also those parts from which pathogens can be carried to the teat during the process of milking. Disinfection of the teat-cups alone is not sufficient.

When the milking unit is carried with the teat-cups hanging downwards, contaminated material is able to run by gravity from the claw and short milk tubes and possibly even the long milk tube into the teat-cups. Furthermore, when a cow is being milked, the milk does not flow constantly in one direction but pulsates up and down the milk tubes. This was demonstrated as follows:—

A milking unit was fitted with glass windows by inserting a piece of glass tubing into each of the 4 short milk tubes, the internal diameter of the glass tubing being the same as that of the milk tubes. One such unit is shown in Fig. 1. A 20-c.c. record syringe was filled with 1 per cent. aqueous methylene blue solution and fitted with a needle. The milking unit was applied to a cow in the normal way and, when the milk flow was established, the needle was inserted into one of the milk tubes, below the glass window. The plunger of the syringe was restrained somewhat so that the dye was sucked into the milk tube slowly.

This procedure was tried on one or more occasions with 8 cows of various ages and stages of lactation, and in every case dye appeared not only at the window just above the needle but at one, two, or all

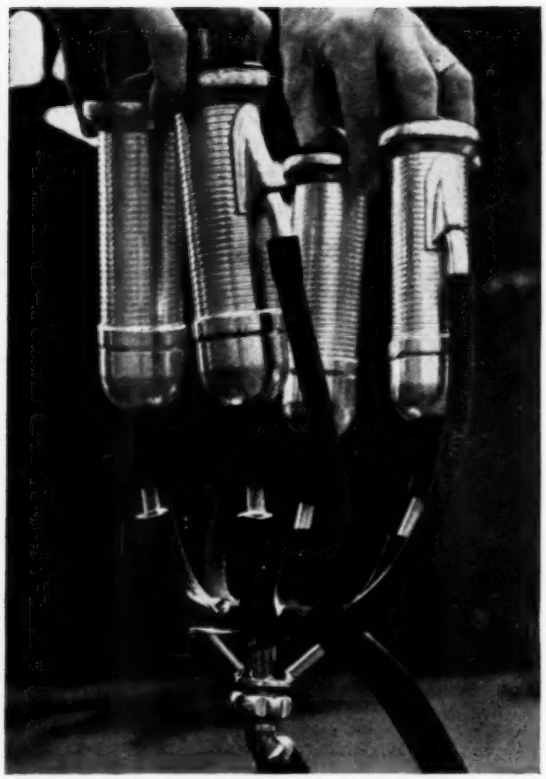

FIG. 1.—Milking unit fitted with glass windows to observe milk flow.

of the others. Even after the needle was removed, columns of dyed milk could be seen rising and falling in the milk tubes synchronously with the pulsation of the machine. The concentration of the dye fell with each pulsation until, after three or four, it was no longer visible. After the machine was removed, the cow's teats were seen to be stained by the dye.

On every occasion when dye was introduced into any of the 4 short milk tubes it passed from one teat-cup to another during milking. This occurred at all stages of milking when the milk was flowing freely. In several cases when one quarter finished milking before the others, dye was no longer drawn up into the corresponding milk tube.

When dye was introduced into the long milk tube nothing was seen in most cases. However, in the case of one cow, when injection was made a few inches from the claw and towards the end of milking, dye regularly appeared at the glass windows in the short tubes.

Thus it has been shown that material can be carried from one part of the milking apparatus to another during milking. The transmission of mastitis infections by the machine can be prevented only if the inner surfaces of the teat-cups, short milk tubes, claw, and long milk tube are sterilised. Although the last item in this list is probably the least important, a contaminated long milk tube clearly gives rise to some risk of infection; a disinfection method which deals with it is superior in this respect to one which does not.

Materials and Methods

Milking Machines. The machines used were of the bucket type fitted with stretched liners whose mouthpieces were formed by the insertion of rubber discs.

Testing of Disinfection Techniques. A comparison of various techniques for disinfecting milking units can be made by contaminating units with milk from cows affected with mastitis by passing equal volumes of such milk through a number of units, which are then disinfected by different methods and the number of mastitis pathogens which survive estimated. This method has the advantage that the same milk can be used to contaminate several units so that several tests can be made on the same day. However, Claydon (1953b) has shown that inconspicuous deterioration of the rubber surfaces can cause different units of the same design to retain different amounts of contamination. Furthermore, in cases of mastitis, the fore-milk is often the most abnormal while, as milking proceeds, the appearance and consistency of the milk become more normal. This sequence of events cannot be imitated using mixed milk from a cow with mastitis. Such milk resembles neither fore-milk, mid-milk, nor strippings, and may not adhere to rubber surfaces in the same way.

For these reasons it was decided that the only satisfactory way to test a disinfection method was to use it on a milking unit which had been used to milk a cow affected with mastitis, even though this method restricts the number of tests that can be made to one per cow per milking. The following procedure was adopted:—

For each series of tests a cow affected in one or more quarters with mastitis caused by *Str. agalactiae* was chosen. This cow was milked on successive mornings with the same milking unit (*i.e.* teat-cups, short milk tubes, claw, and long milk tube). The unit was chosen from one of those in use in the laboratory herd, so that the rubber parts were neither new nor worn out. The unit was sterilised in a steam chest each day before use. On one day, the total number of *Str. agalactiae* remaining in the unit after milking was estimated. On the other days, various disinfection procedures were carried out and the number of *Str. agalactiae* remaining thereafter estimated.

The number of organisms secreted by an infected cow can sometimes vary widely from day to day. As a check on this possibility, a viable count was carried out each day on a sample of the cow's milk taken from the milking bucket.

Some of the cows used had been infected by infusion with a culture of *Str. agalactiae via* the teat canal. Others had become infected by exposure to these artificially-infected ones.

Recovery of Str. agalactiae *from Contaminated Milking Units.* Organisms were recovered by passing 1,000 c.c. of sterile milk (or inactivating agent) through the unit 10 times, using the apparatus illustrated in Fig. 2. The teat-cups were suspended

vertically by inserting a rubber tube with an expanded end into the mouth of each and connecting the four tubes to a milking unit claw. A rubber tube led

FIG. 2.—Apparatus for recovering *Str. agalactiae* from a contaminated milking unit.

from the claw to a milking bucket in which the sterile milk was placed. The contaminated milking unit was connected to a second bucket fitted with operating cover and pulsator and the milk was drawn through the unit by the vacuum pump of the milking machine.

The object of passing the same milk through the unit 10 times was to allow the concentration of organisms in the washings to reach equilibrium with that in the milk left behind (adhering to the inner surfaces of the unit).

Removal of the contaminating material was aided by allowing the liners to pulsate and by allowing air to enter around the mouth of each teat-cup, causing churning of the milk.

All the equipment used in this operation was sterilised by exposure to free steam in a steam chest held at 100° C. for 15 minutes.

When the teat-cup liners were dealt with separately from the other parts of the milking unit, they were incorporated into another milking unit, the remainder of which had been sterilised.

Plate counts of viable *Str. agalactiae* were carried out on the washings, serial 10 times dilutions being made if necessary. At least 3 plates were used for each dilution: up to 10 plates were used if the number of organisms was expected to be small.

Disinfection Techniques Used

1. *Rinsing with water.* Two variations of the technique were used:—

(a) Successive volumes of cold or hot water (sometimes containing detergent) were drawn through the unit using the vacuum pump of the milking machine and allowing the teat-cup liners to pulsate.

(b) Cold water was passed through the unit from a tap.

2. *Circulation of hot water or disinfectant through the unit.* An automatic device sold commercially for washing milking units and operated by the vacuum pump of the milking machine was used. Either water at 70° C. or cold disinfectant solution was allowed to circulate through the milking unit for three minutes. The water was heated in an insulated, thermostatically controlled, electrically heated tank, also available commercially.

The unit was given a brief rinse with cold water before disinfection by attaching the long milk tube to a tap. The unit was given a second similar rinse after disinfection, using a different tap to avoid re-contamination.

3. *Dipping in disinfectant.* The cluster was first plunged rapidly four times into and out of a bucket of clean, cold water and then four times into and out of a bucket of freshly-prepared disinfectant solution at 60° C. diluted to the strength recommended by the manufacturers. The vacuum-release tap on the milking-bucket cover was removed during this operation and the whole cluster was immersed. The water and the disinfectant were thus able to enter the teat-cups, short milk tubes, and claw freely.

Before attempting to recover organisms from the disinfected unit, the long milk tube was replaced by a sterile one.

4. *Filling the unit with disinfectant.* The disinfectant was allowed to remain for 3 minutes. The unit was rinsed with cold water before and after disinfection as in method 3.

Results

The results are presented in Tables I and II.

Table I shows the distribution of *Str. agalactiae* in various parts of the milking unit; (i) immediately after milking and (ii) after rinsing with water by method 1 (a).

Large numbers of *Str. agalactiae* were found in all parts of the milking unit after it had been used to milk an infected cow. After rinsing with running water, although the number of *Str. agalactiae* in each part of the unit was greatly reduced, the organisms were distributed in a different way; on the average there were more left behind on the teat-cup liners than elsewhere. Immediately after milking 53 per cent. of the organisms were on the liners (average of 4 tests); after rinsing 77 per cent. were on the liners (average of 5 tests). This indicates that it is relatively more difficult to remove the organisms from the liners than from other parts of the unit.

It is interesting to note that on one occasion (series 8 in Table II) the concentration of organisms in the washings from the milking unit exceeded that in the sample of the cow's milk taken from the milking bucket after milking. The concentration in the washings was 43,000 per c.c. (*i.e.* 43,000,000 in 1,000 c.c. of washings) and that in the cow's milk 20,000 per c.c. This may have been due at least in part

TABLE I

DISTRIBUTION OF *Str. Agalactiae* IN MILKING UNITS

(i) Immediately after milking

	Teat-cup liners	Short milk tubes and claw	Long milk tube
	66,000,000 24%	66,600,000 24%	143,000,000 52%
	163,000,000 73%	39,400,000 18%	19,800,000 9%
	43,000,000 55%	31,800,000 40%	3,900,000 5%
	102,000,000 61%	44,500,000 27%	19,500,000 12%
Average	53%	27%	20%

(ii) After rinsing with water

	Teat-cup liners	Short milk tubes and claw	Long milk tube
	10,800,000 96%	144,000 1·2%	316,000 2·8%
	473,000 64%	151,000 21%	110,000 15%
	5,400,000 75%	1,490,000 21%	270,000 4%
	5,500,000 64%	2,800,000 32%	340,000 4%
	1,460,000 86%	215,000 13%	18,500 1%
Average	77%	18%	5%

to clumping of organisms in the milk (due to clotted mastitis secretion) causing a falsely low count. However, the very large number of *Str. agalactiae* recovered from the milking units in many of the trials suggests that the organisms may be able to build up in numbers in excess of their concentration in the milk.

Neither the number of organisms in the cow's milk nor the physical condition of the milk had any significant effect on the *distribution* of the organisms either before or after rinsing.

The results obtained with the various disinfection methods are given in Table II.

The accuracy of bacterial counts carried out on mastitis secretion is of necessity low. When the milk contains large clots it is often impossible to ensure an even distribution of organisms in the sample or in the dilutions made from it. In a number of cases replicate plates of the same dilution were so greatly at variance with one another that no estimate of the number of organisms in the sample could be made. Such cases have been recorded as " not countable " in the Table.

On several occasions there was considerable variation in the milk counts from day to day. Even when there was little variation in the milk count it is possible that, in some cases, there may have been a change in the consistency of the milk which affected the number of organisms remaining in the unit. This possibility must be allowed for in interpreting the results. In the few trials in which such changes were obvious the trial was discontinued and excluded from the published results.

In general, counts of washings were much more consistent than those of milk. This is probably because the mastitis secretion was broken up and the organisms more evenly distributed by the process of recovering organisms from the milking unit.

Method 1 (*a*). *Rinsing with water.* (Series 1 to 6 in the Table). This method was devised with a view to its application in milking parlours where the multi-position vacuum tap could be adapted for the drawing of rinse-water through the unit. In all the trials where 3 successive rinses were used, the second rinse was of water at 60° C. All other rinses were of cold water.

The efficiency of this method was markedly affected by the physical condition of the cow's milk. When the milk contained clots only, rinsing with water removed all but about 1 per cent. of the organisms (in four trials the recovery rate ranged from 0.33 to 1.3 per cent.). However, when the fore-milk was of a sticky, purulent nature, much larger numbers of organisms remained after rinsing (5 trials, range 1 to 29 per cent.).

Increasing the amount of water in each rinse from 2 to 4 pints made no difference to the number of organisms removed.

There was some evidence that omission of the warm water rinse reduced the efficiency of the method but this was tried only once (Series 3). In this trial 7.2 per cent. of the organisms remained.

The addition of an inorganic detergent* to the warm rinse water gave somewhat better results, especially when the fore-milk was purulent (Series 5).

Method 1 (*b*). *Rinsing with water from a tap* (Series 15 and 16). This method could be applied in either milking parlours or cowsheds. It was tried only on cows with purulent fore-milk. The recovery rate of organisms after rinsing was 21 per cent. and 57 per cent. respectively in 2 trials.

Method 2. *Circulation of water or disinfectant* (Series 8, 9, and 14 to 16). The application of this method requires the use of 2 milking units for each bucket. One unit can then be disinfected while the other is in use. Since some cows can be milked in 3 minutes, the method must be effective within this time.

The circulation of water at 70° C. through the unit was the most efficient of the methods tested. However, even this method did not achieve complete disinfection; the recovery rate in the 2 trials was 0.012 per cent. and 0.049 per cent. respectively.

The circulation of cold hypochlorite† solution was no better than rinsing with water (recovery rate 1.9 per cent.).

When the fore-milk contained pus, the circulation of a quaternary ammonium disinfectant‡ (which contained an organic detergent in addition to the q.a.c.) was more effective than rinsing with water. The recovery rates of 3.9 per cent. and 18 per cent. show

* Calphos.

† Deosan. ‡ Bactergent. § Hibitane udder wash.

TABLE II
DISINFECTION OF MILKING UNITS

Series	Nature of fore-milk	Number of *Str. agalactiae* per c.c. of milk	Disinfection procedure (Refer to text for description of methods 1, 2, etc.)	Number of *Str. agalactiae* recovered	Number recovered as percentage of those recovered from undisinfected unit
					Per cent.
1	Clots	9,630,000	None	222,000,000	—
	,,	7,400,000	Rinsed with 3×2 pints of water (Method 1a)	734,000	0·33
2	Pus	1,700,000	None	78,700,000	—
	,,	4,000 000	Rinsed with 3×2 pints of water (Method 1a)	7,160,000	9·1
	,,	700,000	,, ,, 3×4 ,, ,, ,, ,,	8,640,000	11·0
3	Clots	10,800,000	None	207,000,000	—
	,,	14,800,000	Rinsed with 3×4 pints of water (Method 1a)	2,690,000	1·3
	,,	Not countable	,, ,, 2×2 ,, ,, ,, ,,	15,000,000	7·2
4	Pus	Not countable	None	166,000,000	—
	,,	,, ,,	Rinsed with 3×4 pints of water (Method 1a)	1,690,000	1·0
5	Pus	5,100,000	None	125,000,000	—
	,,	19,300,000	Rinsed with 3×4 pints of water (Method 1a)	36,000,000	29·0
	,,	Not countable	,, ,, ,, ,, ,, ,, with detergent (Method 1a)	8,300,000	6·6
6	Clots	Not countable	None	830,000.000	—
	,,	271,000,000	Rinsed with 3×4 pints of water with detergent (Method 1a)	7,400,000	0·89
	,,	81,000,000	,, ,, ,, ,, ,, ,, (Method 1a)	10,500,000	1·3
7	,,	220,000	None	14,000,000	—
	,,	225,000	Dipped in water and 1/5,000 chlorhexidine (Method 3)	32,000	0·23
8	,,	20,000	None	43,000,000	—
	,,	110,000	Hot water circulated for 3 minutes (Method 2)	5,000	0·012
9	,,	107,000	None	10,200,000	—
	,,	111,000	Hot water circulated for 3 minutes (Method 2)	5,000	0·049
10	,,	18,600,000	None	180,000,000	—
	,,	17,500,000	Dipped in water and 1/10,000 chlorhexidine (Method 3)	107,000,000	59·0
11	,,	22,200.000	None	750,000,000	—
	,,	19,400,000	Dipped in water and hypochlorite (Method 3)	21,100,000	2·8
12	,,	5,100,000	None	168,000,000	—
	,,	6,750,000	Dipped in water and 1/5,000 chlorhexidine (Method 3)	124,000,000	74·0
13	,,	35,500,000	None	1,480,000,000	—
	,,	17,000,000	Dipped in water and hypochlorite (Method 3)	65,000,000	4·4
14	,,	117,000,000	None	1,120,000,000	—
	,,	138,000,000	Filled with hypochlorite for 3 minutes (Method 4)	29,000,000	2·6
	,,	128,000,000	Hypochlorite circulated for 3 minutes (Method 2)	21,500,000	1·9
15	Pus	45,000	None	10,500,000	—
	,,	9,000	Rinsed with 6 pints of water (Method 1b)	2,200,000	21·0
	,,	Not countable	Q.a.c. (with detergent) circulated for 3 minutes (Method 2)	406,000	3·9
16	,,	Not countable	None	29,000.000	—
	,,	,, ,,	Rinsed with 6 pints of water (Method 1b)	16,500,000	57·0
	,,	,, ,,	Q.a.c. (with detergent) circulated for 3 minutes (Method 2)	5,200,000	18·0

that a considerable proportion of the organisms survived nonetheless.

Method 3. *Dipping in disinfectant* (Series 7 and 10 to 13). Of the two disinfectants used, hypochlorite† gave distinctly better results. The recovery rates after dipping in hypochlorite were 2.8 per cent. and 4.4 per cent. respectively in 2 trials. Chlorhexidine§ was used at two dilutions, 1:5,000 and 1:10,000. At the former dilution the recovery rate was 74 per cent. and at the latter 59 per cent.

Series 7 and Series 12 compare the bacteriostatic with the bactericidal properties of chlorhexidine. In the former trial, organisms were recovered from the disinfected unit using sterile milk, which does not inactivate the disinfectant at this dilution. The apparent recovery rate was 0.23 per cent. However, when egg-yolk broth (Davies *et al.*, 1954) was used instead of milk, the recovery rate was 74 per cent. This shows that, although chlorhexidine is bacteriostatic at a high dilution, its bactericidal efficiency is not high under these conditions.

Method 4. (Series 14). After filling the unit

with hypochlorite and leaving it for 3 minutes the recovery rate was 2.6 per cent.

Discussion

None of the methods tried effected complete disinfection of the milking unit. In a normal milking routine not more than 3 minutes can be allowed for a disinfection procedure and this can be done only if two units are provided for each bucket. If only one unit is provided then only a few seconds are available for disinfection. Apart from the short time available, the adherence of mastitis secretion to the rubber surfaces of the milking unit reduces the efficiency of all methods except that of heat. Disinfectants have to penetrate through this adherent film in order to be effective. Even if a disinfectant can penetrate, it may be inactivated by the organic matter present, especially since the organic matter content of the adherent film is high. The results of rinsing with various volumes of water show that the amount of contamination can be reduced to a comparatively low level in this way, but, when that level is reached, further rinsing has little effect. For these reasons it is perhaps hardly surprising that complete disinfection was not effected and that the most effective method was that of heat (through the medium of hot water).

Although the circulation of hot water was the most effective method, it involves the use of expensive apparatus (the cost of the water heater was £45 and of the automatic washer £5 10s.) and the continuous consumption of electricity at the rate of 4 kilowatts throughout milking. Extra milking equipment is also required. In view of its cost and because even this method did not effect complete destruction of the pathogen, it cannot be recommended for practical use except on farms where hot water can be supplied cheaply.

When the cow was secreting milk which contained sticky, purulent material, the best results were given in most cases by methods using a detergent, but the results of all methods were irregular and often large numbers of organisms survived. It is evident that, when a cow is severely affected in this way, disinfection is extremely difficult and one cannot hope even to approach complete sterilisation.

When the cow was less severely affected, rinsing with running water gave results at least as good as and generally better than treating with disinfectant. It must be emphasised that the disinfectants were tested under ideal conditions. Solutions were freshly prepared, free from organic matter and, when using the dipping method, made up in warm water (at 60° C.). Under practical conditions in the cowshed such conditions might be realised at the beginning of each milking. However, since the same disinfectant solution is used over and over again, organic matter accumulates, the disinfectant is depleted, and the temperature falls as milking proceeds. Consequently these methods are much less effective at the end of milking than at the beginning.

The traditional method of dipping the cluster in disinfectant solution suffers from the further disadvantage that the long milk tube leading from the claw to the bucket is not disinfected. Indeed, unless the air-lock in the unit is released by opening the vacuum-release tap or removing the lid from the bucket, the solution only partly enters the teat-cups and misses the milk tubes and claw completely. Furthermore, during the process of dipping, contaminated material is transferred from the inside of the unit to the outside and thence to the milker's hands. The pathogens in such material may not be completely destroyed by the disinfectant and may be carried from cow to cow by the milker.

Methods which are so uncertain cannot be accepted.

Rinsing with running water does not suffer from any of these shortcomings and, moreover, is economical and easy to apply. It is not claimed that the particular rinsing methods described in this paper are the ideal ones, but that rinsing with water is of itself more effective than treating with disinfectant.

Acknowledgments.—We are indebted to our colleagues Dr. A. W. Stableforth and Mr. P. Stuart for encouragement in carrying out this work and help in preparing the paper.

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CHLORTETRACYCLINE FOR COLOSTRUM-DEPRIVED CALVES

Forty-eight new-born calves exposed to *E. coli* infection were used in an experiment to compare the value of antibiotics with that of colostrum. Thirty-eight were deprived of colostrum. Ten received chlortetracycline (as Aurofac D, Lederle Laboratories Inc.), 10 received chlortetracycline plus penicillin, and 8 received no antibiotics. The 10 remaining calves received 6 pints of colostrum. The antibiotics were given at the rate of 250 mg. per day of each antibiotic for the first 5 days of life, and 125 mg. per day for the remainder of the 3 weeks experimental period. All calves were reared on whole milk throughout the experiment.

Chlortetracycline given alone significantly reduced the mortality rates, whereas penicillin had no effect. Where both antibiotics were given there was a suggestion that penicillin antagonised the beneficial effect of chlortetracycline. The live-weight gain of chlortetracycline fed calves was significantly greater than that of the colostrum fed group.

C. M. E.

Summarised from an article by P. L. Ingram *et al.*, in *The British Journal of Nutrition*, June, 1958.

Current Literature

ABSTRACTS

Studies on Anthelmintics for Cattle, IV. RIEK, R. F., & KEITH, R. K. (1958). *Aust. vet. J.* **34.** 93-103.

This paper records anthelmintic tests on over 200 calves with a water soluble organo-phosphorus compound first prepared by Schrader & Lorenz in Germany, and known variously as Bayer L13/59, " Neguvon " and " Dipterex."

Over 80 per cent. efficiency was obtained against *Haemonchus* at the dose rate of 1 gm. per 100 lb., against *Oesophagostomum* at 2 gm. per 100 lb. and against *Bunostomum* and *Cooperia* spp. at 5 gm. per 100 lb. Treatments at the rate of 5 gm. per 100 lb. when the worms were in the immature stages gave a striking reduction in the numbers of each of the 4 genera mentioned although *Oesophagostomum* younger than 14 days old were not susceptible.

Toxic symptoms amongst some 200 cattle treated in the field in Queensland were negligible, but 2 amongst a batch of 26 calves in Victoria collapsed and 9 more showed temporary discomfort although all subsequently recovered. The authors suggest the drug should be restricted to veterinary use until its side effects are better appreciated.

Studies on Anthelmintics for Sheep. Some Organophosphorus Compounds. GORDON, H. McL. (1958). *Aust. vet. J.* **34.** 104-10.

Tests on 3 organo-phosphorus compounds are reported. The compound Bayer L13/59 (syn. " Neguvon " or " Dipterex ") was very effective against *Haemonchus contortus* at a dose rate 2 gm. per 100 lb. by rumenal injection. Effects against *Oesophagostomum columbianum* were variable, and *Trichostrongylus colubriformis* was apparently unaffected unless the drug passed directly to the abomasum, when the results were good at 3 to 5 gm. per 100 lb. Such doses were quite safe.

The compound Bayer L21/199 (syn. " Asuntol " or " Muscatox ") showed pronounced activity against *T. colubriformis* but the therapeutic index was too narrow for safety. The third compound (Dow ET/57) showed poor anthelmintic properties against the nematodes under test.

P.A.K.

Value of Interrupted Treatments and Preventive Medication in the Control of Intestinal Coccidiosis Caused by *Eimeria necatrix*. DAVIES, S. F. M. (1958). *J. Comp. Path.* **68.** 363-73.

Experimental infections of *E. necatrix* were only partially controlled by sulphadimidine or sulphaquinoxaline given in the drinking water as an interrupted course of treatment (3 days on—2 off—3 on). Better results were obtained when a further period of treatment was employed, giving a schedule of 3 days on—2 off—3 on—2 off—3 on. Using this extended schedule good control was still obtained with lower concentrations of the drugs, namely 0.15 or 0.1 per cent. of sulphadimidine instead of the usual 0.2 per cent., and 0.032 per cent. of sulphaquinoxaline instead of 0.043 per cent., and the author suggests that better control of field outbreaks may follow the use of these lower concentrations in combination with the extended (3—2—3—2—3) schedule.

For the prevention of the disease, medication of the mash with 0.0125 per cent. of Nicarbazin was more effective than the use of 0.01 per cent. of Nitrofurazone. Nicarbazin prevented mortality and the appearance of symptoms, allowed a steady weight gain and did not prevent the development of resistance to re-infection.

E. A. G.

Occupational Hazards in the Bone-Meal Industry. LAMB, R. (1958). *Lancet*. July 19th, 1958. 151-3.

Bones imported from India, Pakistan and the Argentine are used in the production of animal charcoal for sugar refining. The raw bones are broken in crushing mills and then sorted into three grades of granularity before being reduced to charcoal in ovens. The very fine dust is sold as bone fertiliser. Face masks are used by the workers, but the fine bone dust penetrates everywhere, and in spite of protective clothing it reaches the workers' underclothing. Frequent sampling has shown the presence of virulent anthrax spores in the raw bones, the crushed bones and the bone dust. Four cases of cutaneous anthrax in workers are described, their occupations were labourer, lorry driver, sack repairer and process worker. All recovered following treatment with penicillin.

Two cases of empyema in process workers are described. Investigation showed that these were of clostridial origin, *Cl. tertium* and possibly *Cl. oedematiens*. It is suggested that infection occurred by inhalation, and it is pointed out that if this suggestion is correct it is remarkable that no cases of pulmonary anthrax have occurred in this factory. Both cases of empyema ultimately recovered after treatment with oxytetracycline.

Anthrax and Bone-Meal Fertiliser. GREEN, D. M. & JAMIESON, W. M. (1958). *Lancet*. July 19th, 1958. 153-4.

These authors reported 6 cases of human anthrax in 1955 and then expressed the view that bone-meal used in this country should be rendered safe from the hazards of anthrax. In 1957 the Minister of Health set up a committee of enquiry to consider the existing legal precautions and to make recommendations.

Four more cases of anthrax have been seen since 1955. One patient was a veterinary surgeon who contracted anthrax through contact with an infected animal. The other 3 gave histories of having handled bone fertilisers. *Bacillus anthracis* was isolated from samples of bone fibre handled by one of them. All cases recovered following antibiotic treatment.

In an addendum to the article, there are given brief details of a case of cutaneous anthrax in a seedsman aged 17 years. *B. anthracis* was demonstrated in a sample of bone meal handled by him.

G.F.B.

News and Comment

THE ESSEX VETERINARY SOCIETY

The President of the Essex Veterinary Society, Mr. W. F. Barton of Brentwood, and Mrs. Barton held a reception of members at the Saracens Hotel, Chelmsford on October 24th. Forty-two members and their wives were present.

The date also marked the retirement of Mr. W. T. MacGregor as the Ministry's D.V.O. In a short speech Mr. Barton recalled that Mr. MacGregor had been the first President of the Essex Veterinary Society and he wished Mr. and Mrs. MacGregor long years of health and happiness in their retirement: a cheque was presented to them from the Society and from the L.V.I.s. of Essex. Mr. MacGregor responded, and also introduced to the Society his successor, Mr. A. C. L. Brown.

A GIFT HORSE

An article under the somewhat dramatic title, "Is Your Dog Protected Against the Hard Pad Horror" was published recently in a popular Sunday newspaper. Several veterinary surgeons found the tone of the article regrettable—to say nothing of the writer's confusion between a serum and a vaccine—but in the event they proved to have looked a gift horse in the mouth. In one practice at least the veterinary surgeons have been inundated with requests to carry out vaccinations ever since the article appeared. So effective has it seemed to be, indeed, that we are tempted to wonder whether the titles in this journal, and the style of its presentation, are not too sedate by half.

PERSONAL

Mr. W. T. MacGregor, Divisional Veterinary Officer of Chelmsford, retired on October 23rd, 1958.

Mr. E. Forrest, has been appointed Veterinary Officer and has taken up station at Kendal.

Births

GUILBRIDE.—On October 16th, 1958, at Old Entebbe, Uganda, to Mary (née Winch), wife of Patrick Guilbride, B.SC., M.R.C.V.S., a daughter, Dorothy Lys.

MCKEAN.—On October 6th, 1958, to Isabel, wife of Hugh McKean, M.R.C.V.S., of 3 Albert Street, Lytham, a son, Derek.

COMING EVENTS

November

5th (Wed.). Ordinary General Meeting of the Sussex Veterinary Society at the Old Ship Hotel, Brighton, 2.30 p.m.

6th (Thurs.). General Meeting of the Central Veterinary Society at the Royal Veterinary College, 6 p.m.
Meeting of the Shropshire Veterinary Club at the Oak Hotel, Shelton, Shrewsbury, 8 p.m.

7th (Fri.). Autumn Meeting of the Eastern Counties Veterinary Society at the Angel Hotel, Bury St. Edmunds, 10.45 a.m.
General Meeting of the Yorkshire Veterinary Society at the Hotel Metropole, Leeds, 2.30 p.m.
Annual General Meeting of the Royal Veterinary College Association, 5 p.m., followed by the Commemoration Ball in the evening.

12th (Wed.). Meeting of the Southern Counties Veterinary Society at the Red Lion Hotel, Salisbury, 7.30 p.m.
Annual General Meeting of the West of Scotland Division at the Veterinary Hospital, Bearsden Road, Bearsden, Glasgow, 2.30 p.m.

14th (Fri.). General Meeting of the Midland Counties Veterinary Association at the Birmingham Medical Institute, 36, Harborne Road, Birmingham, 15, 2.15 p.m.

20th (Thurs.). Joint Meeting of the Sussex Veterinary Society and the Mid-Sussex Division of the B.M.A. at the Dudley Hotel, Brighton, 8 p.m.
Annual Dance of the V.V.B.F. County of Ayr Ladies' Guild in Western House, Ayr.

27th (Thurs.). Annual Dance of the V.V.B.F. North of Scotland Division in the Northern Hotel, Aberdeen, 7.30 p.m.
Meeting of the Essex Veterinary Society at the Essex Institute of Agriculture, Writtle, Essex, 7.30 p.m.

BRITISH VETERINARY ASSOCIATION QUARTERLY MEETINGS IN LONDON

Wednesday, November 19th—No. 7, Mansfield Street

10.15 a.m. Finance Sub-committee.
11.45 a.m. Parliamentary and Public Relations Committee.
2.00 p.m. Small-Animals Committee.
4.00 p.m. Farm Livestock Committee.

Thursday, November 20th—No. 7, Mansfield Street

10.00 a.m. Veterinary State Medicine Committee.
11.30 a.m. Home Appointments Committee.
2.15 p.m. General Purposes and Finance Committee.

Friday, November 21st—Connaught Rooms

10.15 a.m. Council Meeting.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Northants. River Meadow, Wharf Road, Higham Ferrers, Wellingborough (Oct. 24).
Sussex. Montague Farm, Wisborough Green, Billingshurst (Oct. 23).
Wilts. Manor Farm, Donhead St. Mary (Oct. 27).

Fowl Pest

Berks. Purley Park Poultry Farm, Reading (Oct. 24).
Essex. Hoggs Farm, Harlow Common, Harlow (Oct. 21); Blacklegs Farm, Great Leighs, Chelmsford (Oct. 25).
Hunts. High Ferry, Chequers Street, Fenstanton, Huntingdon; 21, London Street, Godmanchester (Oct. 22); The

Chequers, Fenstanton; Elma, Chequers Street, Fenstanton (Oct. 23).
Kent. Ivy House, South Street, Meopham, Gravesend (Oct. 23).
Norfolk. Waite Farm, Saham Hills, Shipdham, Thetford (Oct. 21); Corner Ways, Necton, Swaffham (Oct. 23); The Old Rectory, Necton; Mistletoe Farm, Wendling, Dereham (Oct. 24); Hurst Farm, High Street, Marsham; The Laurels, Hickling, Norwich (Oct. 25); 31, Ketts Hill, Necton, Swaffham (Oct. 27).
Worcs. Yarnold Lane Farm, Bournheath, Bromsgrove (Oct. 24).
Yorks. Church Farm, Birkin, Knottingley (Oct. 23).

Swine Fever

Essex. Hude Farm and Kirby Farm, Duton Hill, Great Easton, Dunmow (Oct. 24).
Hants. Tidworth Gardens, Tidworth (Oct. 27).
Kent. 128, Jackson Road, Bromley (Oct. 27).
Lancs. 242, Glazebrook Lane, Rixton, Warrington (Oct. 23).
Norfolk. Lyndhurst, Jubilee Lane, North Lopham, Diss; Ivy Road, Spixworth, Norwich (Oct. 24); Sheringham Road, West Beckham, Norwich (Oct. 27).
Sussex. Cisswood and Field at Prongers Corner, Lower Breeding, Horsham (Oct. 23).
Yorks. Chatt House, Burton Pidsea, Hull (Oct. 21).

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

Following the Articles of Association

SIR,—I was, unfortunately, unable to be present at the Annual General Meeting at Douglas, but in your report on it in THE VETERINARY RECORD, I noticed that the recommendation of the Council regarding the Congress for 1960 was not accepted.

Mr. Beynon suggested that the recommendation was not unanimous, but I would suggest that to expect every decision of the Council to be unanimous is a counsel of perfection, and indeed Article 72 clearly allows for a majority decision. Mr. Wilkinson obviously felt deeply on the matter, and later drew attention to it, but his voice was overruled.

The matter is probably not one of really vital importance, but nevertheless Articles of Association are means by which an association is governed, and if they are to be abrogated in this somewhat light-hearted fashion without the general view of the members being ascertained, then a precedent can be created which could have very far reaching results.

Yours faithfully,
A. J. BEESON.

The Stone House,
Boughton Monchelsea,
Maidstone, Kent
October 22nd, 1958.

"Tranquillising" for Shows

SIR,—I am at a loss to understand Mrs. Robinson's letter. To the best of my knowledge there are no rules of the Kennel Club debarring the use of these drugs. I think, therefore, that prescribing "tranquillising" drugs to highly strung dogs, prior to their being subjected to the show ring with its attendant rigours, is no more iniquitous, or meriting the term doping, than doing the same thing on Guy Fawkes night.

Yours faithfully,
E. L. CASPARI.

Whyteleafe,
Leatherhead.
October 25th, 1958.

Progressive Muscular Dystrophy in the Dog

Sir,—A recent case of pyometra in an 8-year-old Alsatian bitch called to mind an article by J. C. Whitney in THE VETERINARY RECORD of July 26th.

In it 3 cases of muscular dystrophy are described, one in a dog and 2 in bitches. It is interesting to note that in all 3 there were heart lesions, and in the case of the bitches pyometra as well. The writer points out that muscular dystrophy is usually associated with vitamin E. deficiency in cattle and sheep, and he goes on to state: "there was no opportunity to assess the effect of vitamin E. therapy in any of these dogs but it is unlikely that it would have proved beneficial."

It has long been our custom to treat diseases of the heart and circulation in dogs and cats with large doses of vitamin E., a form of treatment which we have found highly satisfactory over the past 13 years. Recently we have used vitamin E (the succinate form of Alpha Tocopherol) on a wider scale both *per se* and as an adjunct to other forms of therapy. It has been particularly useful in heart cases where there is an associated metritis or pyometra. Some cases of pyometra become completely resolved, and where ovariohysterectomy becomes necessary the bitches appear to stand up to the operation much better if a pre-operative course of vitamin E has been given.

In the case of the Alsatian mentioned above, operation was deferred rather longer than usual on account of the difficulty in establishing a diagnosis. X-ray examination did not help, probably due, as we afterwards found out, to the unusually liquid state of the uterine contents. This bitch was treated with antibiotics and 200 units vitamin E daily pre-operatively. In spite of being extremely weak when operation was undertaken, she made a rapid and complete return to normal health and activity.

One of the symptoms noted in this case was dragging of the hind legs, which could not be attributed to a general weakness as it was noticed in the early stages when the bitch was strong and in good bodily condition.

In view of this and many other successful results in cases showing evidence of suspected muscular dystrophy, both in dogs and bitches, we believe that vitamin E, especially if used in large doses, would have been of benefit in the cases described by Whitney.

Yours faithfully,
N. H. LAMBERT.
EILEEN PARKHILL.

47, South Richmond Street,
Dublin.
October 25th, 1958.

The Biologically Active Isomers of Glycyrrhetinic Acid

SIR,—We have read with interest the article under this title and are pleased to note that our preliminary impressions (*Vet. Rec.* **69.** 573) have now been confirmed statistically in controlled trials.

Recently we have used a new derivative of glycyrrhetinic acid, namely, the disodium salt of glycyrrhetinic acid hydrogen succinate (Biosone G.A. brand) in the form of an intra-articular injection, and would like to report our preliminary results:—

Subject: Alsatian, 6-year-old, dog.

History: The right hind leg had been lame for 3 months. Examination showed a very swollen hock joint, hard, painful, grating when moved. There was considerable wasting of leg. X-ray examination showed considerable arthritic damage.

Treatment: The dog was anaesthetised, 1½ c.c. of fluid was withdrawn from the joint and replaced by 1½ c.c. Biosone G.A. brand disodium salt of glycyrrhetinic acid hydrogen succinate 25 mg. per c.c. The technique was adapted from that described in THE VETERINARY RECORD (*Vet. Rec.* **69.** 659).

Results: Next morning the joint was swollen and very painful and the dog was unable to put the foot on the ground. The pain and swelling gradually disappeared. After 7 days the dog was walking comfortably, and after 3 to 4 weeks no pain was evident on manipulation. The dog was using the leg naturally. Six weeks later the dog was seen again and an X-ray taken. This showed a substantial reduction in the inflammatory processes and the joint looked much healthier but not entirely normal. The dog was still walking with a slight limp probably due to the impediment of the still swollen joint. Considerable regeneration of muscle was noticed.

Subject: Boxer, 3-year-old, bitch.

History: Hind leg had been sprained. X-ray examination showed very distended synovial protrusions of the right hock joint. The bitch was in great pain and there was substantial wasting of the leg muscles.

Treatment: The bitch was anaesthetised and 3½ c.c. synovial fluid was extracted and replaced by 3½ c.c of the Biosone G.A. intra-articular injection as used in Case 1.

Results: As in Case 1, the following day the joint was very swollen and painful. Improvement has been gradual. When seen a few days ago, *i.e.* 4 weeks after injection, the bitch was still lame but markedly improved. All the soft synovial swelling had disappeared.

Further work is obviously needed, but we feel that these cases may be of interest to other veterinarians and that the results give further evidence of the powerful anti-inflammatory properties of this brand of glycyrrhetinic acid.

Yours faithfully,
J. J. DAVIES,
A. FAIRWEATHER.

1 Church Street,
West Ham,
London, E.15.
October 21st, 1958.

Induction of Anaesthesia using Pentobarbitone Intrathoracically

SIR,—While the blind introduction of barbiturates into the thoracic cavity and into the peritoneal cavity may be necessary on occasions the more accurate intravenous route is preferable for routine use, since the depth of anaesthesia can be assessed during the administration of the barbiturate. Pentothal sodium, which will give a satisfactory duration of anaesthesia for spaying, may then be used, avoiding the necessity of retaining the cat overnight.

The technique I have found most suitable is to use a 4½ × 13 mm. needle with a short bevel and an eccentric 5 c.c. syringe. The skin over the vein is carefully clipped and swabbed with an excess of methylated spirit. A length of rubber tubing is applied as a tourniquet above the elbow and fastened by a pair of Spencer Wells artery forceps.

If the paw is now held in the left hand and the leg pulled forward the vein can be seen very clearly and the needle is guided into the vein. After blood has been withdrawn into the syringe it is held in the left hand while the right hand releases the artery forceps holding the tourniquet.

This method is useful if no assistance is available, and provided a sharp needle is used venipuncture in the cat presents fewer problems than in the dog. I was shown this method a number of years ago and can recollect few occasions when it has been unsuccessful.

Yours faithfully,
G. R. ROBBINS.

Royal Veterinary College,
Beaumont Animals' Hospital,
Royal College Street,
London, N.W.1.
October 22nd, 1958.